

Human Larva Migrans Syndrome

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Some animal nematodes are capable of starting life cycles in mammals that are not their definitive hosts, but cannot complete them. The aberrant migration of the larvae may cause severe disease. Animal nematodes causing human larva migrans in the United States are illustrated in Table 1.

In 1952, *Toxocara canis* larvae were reported in the tissues of children with eosinophilia- hepatomegaly and the term visceral larva migrans was proposed.¹ Today, *Toxocara canis* is the principal cause of visceral larva migrans and less commonly *Toxocara cati*. Because the larvae cannot mature, they tend to migrate for months in various tissues until they are overcome by the inflammatory reaction of the host. Toxocarosis, formerly toxocariasis, may also be used for the syndrome. Occasionally, other nematodes, such as *Baylisascaris procyonis*, may be responsible for larva migrans syndrome. *Ancylostoma* species (hookworms) are capable of causing cutaneous larva migrans which is also known as creeping eruption in people.

People generally acquire larva migrans by ingesting *Toxocara* embryonated eggs in the soil. They may also acquire *Toxocara* larvae from unwashed hands, consumption of raw vegetables and consumption of undercooked tissue of infected paratenic hosts such as chickens, cattle and sheep.² Direct contact with infected animals is not a major source of infection because of the incubation period (minimum of two weeks) required for the eggs to become infective. A history of eating soil, common in toddlers, increases the risk. Cutaneous larva migrans is acquired by contact with soil contaminated with *Ancylostoma* larvae.

Prevalence

Human larvae migrans syndromes are not reportable in California, except under the occurrence of unusual diseases. It is under recognized, but the development of a sensitive and specific serologic test has

stimulated clinical and epidemiologic research on larva migrans. The extent of tissue injury is directly proportional to the number of eggs ingested and the number of viable larvae that enter the tissue. Currently, an enzyme-linked immunosorbent assay (ELISA) is the most widely used test. Seroprevalence of toxocarosis in children in the United States was found to be 7.3%.³ Seroprevalence is higher among some groups of socioeconomically disadvantaged children. However, most people do not develop overt clinical disease. The value of immunodiagnostic tools is dependent on the positive predictive value of the test used, which in turn depends on the prevalence of disease in the community.⁴ Since 1994, in Los Angeles County, five cases of visceral larva migrans have been reported. All were Hispanic children with eosinophilia and ELISA titers of 1:32 or higher.

The diagnosis is considered in people with persistent eosinophilia. Identification of larvae in biopsy tissue permits the definitive diagnosis, but biopsy is often unrewarding. In people with clinical signs and history consistent with visceral larva migrans, an ELISA titer of 1:32 or more is considered diagnostic. Toxocaral titers may remain el-

evated for years and a measurable titer is not proof of a causal relation between *Toxocara* and current illness.

Treatment of asymptomatic toxocarosis is not recommended. Treatment of individuals with clinical signs is primarily supportive and anthelmintic therapy is controversial.^{5,6}

Visceral versus Ocular Migration

In children under five years of age, visceral migration through the liver, lungs, and brain is common while in older children the eyes are most often involved.⁶ The reason for this is not known. Some speculate that lower doses of *Toxocara* larvae are associated with higher probability of ocular migration. The ocular lesion, typically unilateral, resembles retinoblastoma. It was first reported in 24 eyes that had been enucleated, in most instances after a clinical diagnosis of retinoblastoma.⁷ A third syndrome called covert toxocarosis is reported in children with cough and wheeze with a history of headache and abdominal pain.^{2,8}

Raccoon Roundworms

Baylisascaris procyonis, an intestinal
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Table 1. Animal Nematode Larva in People

Parasite	Natural Host	Transmission to People
<i>Toxocara canis</i>	Dogs and other canids	Ingestion of embryonated eggs*
<i>Toxocara cati</i>	Cats and other felines	Ingestion of embryonated eggs
<i>Baylisascaris</i> spp	Raccoons and wild mammals	Ingestion of embryonated eggs
<i>Ancylostoma</i> spp	Cats and Dogs**	Skin penetration by larvae

* eggs become infective after about a month on the ground and may remain infective for months.

** *Ancylostoma* spp have been detected in local coyotes.

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roundworm of raccoons, is prevalent throughout North America. Other species of *Baylisascaris* are found in skunks, woodchucks and bears. This species is capable of infecting people and other animals. It is much more pathogenic as the larvae, unlike *Toxocara*, grow larger as they migrate and tend to invade the central nervous system.

With livestock, hay, other foodstuffs and bedding materials contaminated with raccoon feces are a hazard. Ground-fed birds such as doves and pigeons are at increased risk. At the Los Angeles Zoo, feral raccoons defecating in animal compounds can be a problem. Most animal and human cases are identified at necropsy.

How Cats and Dogs Acquire Toxocara

Most puppies become infected with *Toxocara* in utero. One study found as many as 15 per cent of all adult dogs infested. ⁹

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Table 2. How to Avoid Larva Migrans

1. All pet cats and dogs should receive anthelmintics as indicated. It is important to treat bitches and puppies several times after whelping to prevent the passage of infectious eggs.
2. Parents should not allow children, especially those with pica, to play unattended outdoors where they are likely to have access to infectious eggs.
3. Laws prohibiting puppies and dogs from running free and defecating in public areas, particularly areas used by children should be enforced.
4. Animal feces should be kept out of children's sandboxes and play areas.

However, roundworm eggs may not be present in the stools of all infected dogs. Puppies and lactating bitches are most likely to pass infectious eggs.

Toxocara canis of dogs and *Toxocara cati* of cats can be acquired by ingestion of: eggs, paratenic hosts (birds, mice, etc.), infected milk and vomitus or feces of infected youth. With *Toxocara canis*, puppies may also acquire infection by transplacental migration. In pregnant bitches, dormant roundworm larvae are activated and migrate to the liver and lungs of unborn puppies. After birth, the larva complete their migration to the intestine and develop into adult worms in four-month-old puppies. Milk-borne infection during nursing is a major source of roundworms in kittens.

Treatment of Roundworms in Cats and Dogs

Because puppies are often born infected with *T. canis* due to transplacental migration, treatment is recommended at two weeks of age, before eggs are first passed in the feces, and repeated at 4, 6 and 8 weeks to kill all worms. Most puppies are first seen by a veterinarian at six to eight weeks of age. These puppies should be treated twice for roundworms at two week intervals for at least three treatments.¹⁰

There are several anthelmintics that are effective against roundworms.¹⁰⁻¹² One of the more widely used is pyrantel pamoate which is effective, safe and is active against both roundworms and hookworms. Other anthelmintics effective against roundworms

and hookworms include: fenbendazole and mebendazole. Although not approved for use in cats, pyrantel pamoate has been reported to be effective in cats. Lactating bitches can be treated 2-3 weeks after whelping with pyrantel. Preparturient bitches can be treated with fenbendazole. Combinations of anthelmintics are also used to create broad spectrum wormers.

Prevention of Larva Migrans Syndromes

Prevention is aimed at reducing the opportunities for children to ingest soil contaminated by feces of cats, dogs, and other animals. *Toxocara* eggs need a minimum incubation period of two weeks under the right conditions before they become infective. Several weeks may be required before eggs become infective. Direct sunlight and desiccation are rapidly lethal to eggs, but in humid soil they may survive for months or even years. Most cases of human toxocarosis are prevented by: careful personal hygiene, elimination of intestinal parasites from pets, and not allowing children to play in potentially contaminated environments. Ways to avoid larva migrans are illustrated in table 2.

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